Reticulate Hyperpigmentation of Iijima, Naito and Uyeno

A European Case

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A 15-year-old girl is presented with symmetric, hyperpigmented streaks and whorls on trunk and extremities. The pigmentation is located in the basal layer of the epidermis. The clinical and histopathological picture seems to be identical to the reticulate hyperpigmentation of Iijima, Naito and Uyeno, hitherto only described from Japan. Key word: Epidermal melanosis.

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Iijima, Naito and Uyeno have reported two cases of “reticulate hyperpigmentation distributed in a zosteriform fashion, a new clinical type of hyperpigmentation” (1). The patients were little girls; 4 months and 5 years of age, respectively. The younger one had since 2½ months numerous light brown pigmented spots, 1-2 mm sized, gradually spreading on her body except for the face, palms and soles. The spots formed by coalescence areas of hyperpigmentation in a linear and zosteriform distribution. The elder girl had 2-3 mm sized light brown spots, beginning at the age of 2 years, on the inside of both lower legs, gradually spreading to thighs, dorsa of the hands and finally to the abdomen. In neither case were family members affected. Eosinophils were increased 13% and 17%, respectively. Examination of skeleton and eyes showed no abnormalities. Histopathological examination showed increased basal pigmentation, but no increase of melanocytes and no pigmentary incontinence.

The authors compared their two cases with two other cases previously reported in the Japanese Journal of Dermatology in the Japanese language.

The age of onset, the size of the spots, their colour, distribution, slight eosinophilia and histopathological findings were the same. However, the spots were described as depressed.

Progressive cribriform and zosteriform hyperpigmentation (PCZH), as described by Rowr et al. (2) is somewhat similar to the four Japanese cases. The age of onset in these 5 cases was higher; 10-18 years. Hyperpigmented spots, coalescing into a zosteriform arrangement, were seen only on the torso or thighs and limited to one dermatome. Histopathological findings were similar to the Japanese cases.

Iijima et al. consider reticulate hyperpigmentation distributed in a zosteriform fashion to be a generalization of PCZH.

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Fig. 1. Distribution of pigmentation in our case.
CASE REPORT

A Caucasian girl was born 1972, healthy and of normal weight after an uncomplicated pregnancy. Her skin was normal. No blisters, papules, erythema or pigmented lesions were seen, except for a 7x10 mm naevus on her abdomen. It was diagnosed as naevus spilus (3). At the age of 3 years a woolly hair naevus (4) was observed on the right side of the scalp forming a couple of 2-3 cm patches.

In October 1987 she was referred to the Department of Dermatology in Malmö because of mild acne. Hyperpigmentation of several parts of her body was revealed. She claimed that she had discovered it two years before, while taking a shower. She had no previous symptoms or signs, no change in shape, arrangement or distribution. She did not remember having used any drugs, ointments or cosmetics before onset. She had not exposed herself to the sun before or after the discovery. There was no pigmented disturbance in the family, or any history of woolly hair, atopy or other diseases of the skin. The skin changes were light brown spots sized 2-3 mm, irregular in shape with clearly defined borders. They were coalescing to streaks and whorls symmetrically on the trunk, arms and legs (Figs. 1-3).

Histopathological examination revealed a normal epidermis. At the base of the rete ridges, the cells showed an increase of granular pigment in the cytoplasm, but no increase of melanocytes. No melanophages were seen in the corium. Blood-ESR, Hb, leukocytes and eosinophils were normal. As she had not exposed herself to the sun, it is not known whether the lesions will be affected by it or not. The clinical course is hitherto unchanged. No treatment has been given.

DISCUSSION

In our case the shape of the pigmented spots, the arrangement and distribution of the symmetric streaks and whorls, as well as the histopathological findings correspond to the cases of Iijima et al. One of their cases also had a naevus spilus. As in their cases, there has been no inflammatory stage and no pigmented incontinence, thereby excluding incontinencia pigmenti. Otherwise the distribution of the pigmentation with whorls and streaks resembles that disease. There are no nevotuberos or depigmented macules suggesting Morbus Recklinghausen, nor amyloid deposits indicating macular amyloidosis. The distribution of pigmentation was neither depressed nor acral as in acral reticular hyperpigmentation of Kitamura (5).

The lack of epidermal hyperplasia, both clinically and histologically, excludes epidermal naevis including ichthyosis hystrix and inflammatory linear verru-
Crusted Scabies in Acquired Selective IgA Deficiency

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Crusted scabies, an unusual clinical variant of human scabies mite infestation, is usually reported in cases of gross debility, mental deficiency, or immunosuppression. We report here the occurrence of crusted scabies in a 40-year-old man with acquired selective IgA deficiency suspected to be caused by long-term medication with phenytoin for epilepsy. Key words: Mite infestation; Immunosuppression; Epilepsy.

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Crusted scabies is caused by overwhelming infestation of Sarcoptes scabiei (1-3). Several factors are believed to be of importance in its development - mental deficiency, gross debility and lack of skin sensation are traditionally implicated (1). Other authors speculate on the role of vitamin A (4). In addition, the involvement of immune suppression in some diseases (5, 6) and iatrogenic mechanisms such as immunosuppressant (1, 7) or steroid therapy (8) have been frequently discussed.

Hancock & Ward (9) reported a correlation between scabies infestation and low serum levels of IgA, the predominant antibody component of external secretions. They postulated that reduced IgA secretion due to low serum IgA levels may predispose to scabies infestation.

We report a case of crusted scabies occurring in a patient with acquired selective IgA deficiency suspected to be caused by epilepsy treatment with phenytoin.

CASE REPORT

A 40-year-old man with epilepsy was referred to our clinic, suffering from widespread, slightly pruritic hyperkeratotic or crusted eruptions of 2 months' duration. Ten months previously he had been admitted to another hospital, suffering from cholestatic liver dysfunction. Though the course of liver dysfunction was good, the patient developed pruritic eruptions interdigitally and which extended to the trunk. Thickened scaly lesions then developed.

Clinical features showed numerous brownish crusted papules on the trunk and extremities. The palms and soles were grossly hyperkeratotic with large scales. A marked feature was the hyperkeratotic or crusted plaques, particularly on the finger webs, back and buttocks (Fig. 1). Scrapings from hyperkeratotic lesions disclosed numerous mites. A punch biopsy specimen obtained from the buttock showed portions of mite body in the stratum corneum and a mild, non-specific inflammatory cell infiltration. The patient had had epilepsy, for which he received periodic psychiatric treatment. For 10 months, he had been medicated with phenytoin (600 mg/day). His intelligence was deemed slightly subnormal.

Laboratory data included the following (normal range in parentheses). Serum immunoglobulins: IgG, 1020 mg/dl (1000-1700 mg/dl); IgA, 1.0 mg/dl (90-330 mg/dl); IgM, 79